ABSTRACTS OF PRIZE-WINNING PAPERS

FOURTH ANNUAL PRIZE CONTEST

CLINICAL SOCIETY OF THE NEW YORK DIABETES ASSOCIATION, INC.

*The Impaired Carbohydrate Metabolism of Chronic Uremia: Effects of Potassium**

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IMPAIRMENT of carbohydrate tolerance has been noted in chronic uremia. Normal persons experimentally depleted of potassium have also been shown to develop impaired carbohydrate tolerance. The effect of the administration of oral potassium on the carbohydrate abnormalities of uremia was investigated.

Five patients with chronic stable uremia were studied. Total exchangeable body potassium, measured with ^{42}K, was reduced in all, although serum potassium levels were normal or elevated. Each patient was studied under conditions of controlled diet and activity. Oral glucose (OGTT), intravenous glucose (IV-GTT), intravenous tolbutamide (TTT), intravenous insulin (ITT), glucagon (Glu-TT), and epinephrine (ETT) tolerance tests were performed. Also measured were blood urea nitrogen (BUN), serum creatinine and electrolytes. Following the control period, supplemental oral potassium was administered; rigorous care was taken to avoid potassium intoxication. Following the return of total body potassium to normal levels, all studies were repeated.

The OGTT and TTT, initially diabetic in all patients, reverted toward normal following potassium administration. Immunoassayable insulin values during OGTT and TTT rose, with somewhat earlier peak

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levels, following potassium. Little change was noted with IV-GTT. ITT, ETT, and Glu-TT, initially normal, remained unaffected.

In most patients BUN fell during potassium supplementation but in none did serum creatinine change. Thus, improvement in carbohydrate tolerance was dissociable from change in renal function. Since no resistance to exogenous insulin was observed, improved OGTT and TTT appear related, at least in part, to the improved insulin response after potassium supplementation.

*The Influence of Insulin on the Transport of Isonicotinic Acid Hydrazide into the Perfused Liver**

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OUR previous investigations have shown that transport of several drugs across some cell membranes is insulin-sensitive. A study has been made of the effect of insulin on the transport of isonicotinic acid hydrazide (INH) into the liver, a tissue which is considered to be insensitive to the hormone with respect to glucose penetration.

The Miller technique was used to perfuse liver of fasting male Wistar rats weighing 250 to 400 gm. INH was determined by the Deeb and Vitagliano method. Perfusions were conducted for 45 and 90 minutes, and the volume of perfusate, which consisted of heparinized rat blood was 90 to 100 ml.

The single administration of insulin *in vitro* (0.01 U./ml.) and also the continuous infusion of insulin (0.01 U./ml./90 min.) caused an increase of the INH level in the liver at 15 min., after which there was no difference (at the end of perfusion) in tissue concentration of the drug due to insulin. The rate of destruction of INH was not affected. However insulin more than doubled the elimination of INH in the bile. The difference in biliary excretion of INH was of the same order of magnitude as the difference in the total liver content at 15 min.; this suggested that the biliary effect may be secondary to an increase in the penetration of the drug into the hepatic parenchyma.

These results show that the liver cell membrane is insulin-sensitive with respect to drug transport.

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*The Effect of Feeding Frequency on Plasma Insulin and Growth Hormone**

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THE effect of frequency of feeding an isocaloric diet on blood glucose, plasma insulin, and plasma growth hormone was studied. A formula diet was fed in three meals on one day and in 20 half-hourly feedings on another day to five male and five female subjects.

Over-all insulin output over a 13-hour period was almost identical for the two regimens and was similar in males and females.

Over-all plasma growth hormone levels were several times higher in females than in males. The number of specimens with significantly elevated levels of growth hormone was higher during meal feeding than during continuous feeding and, in females, the total growth hormone output was higher during meal feeding than during continuous feeding. Whereas a clear cyclical pattern in growth hormone levels could be discerned during meal feeding with peaks of hormone occurring in the late postabsorptive phase, similar albeit less pronounced peaks occurred during continuous feeding. These data suggest a pattern of growth hormone release independent of negative glucose concentration feedback. This cycling may be related to previously accustomed feeding schedules or to some other central nervous system mechanism.

There was no correlation between total secretion of growth hormone and total secretion of insulin, in that subjects with highest levels of growth hormone did not necessarily have high levels of insulin.

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